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Deep venous thrombosis after myocardial infarction
Predisposing factors

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Of 89 patients admitted to coronary care after myocardial infarction, 27 per cent developed radioisotopic evidence of deep venous thrombosis. This complication occurred more frequently if the patient was over 60 years, if there was a previous history of angina pectoris, and if heart failure or a significant dysrhythmia developed. A group of patients with a low incidence of deep venous thrombosis was also identified.

It has been established by the use of radioactive fibrinogen that deep venous thrombosis of the lower limb occurs in approximately one-third of patients admitted to hospital with acute myocardial infarction (Nicolaides *et al.*, 1971; Murray *et al.*, 1970; Maurer, Wray, and Shillingford, 1971; Simmons, Sheppard, and Cox, 1972; Handley, Emerson, and Fleming, 1972).

The aim of this study is to identify those patients who are especially likely to develop deep venous thrombosis.

Patients and methods

Patient selection Ninety-four patients were selected for study. All were admitted to the coronary care ward at Bradford Royal Infirmary. The admitting officers assessed the clinical features and admission electrocardiogram of the patient. If they decided that a diagnosis of myocardial infarction was probable, and if a supply of fibrinogen labelled with ^{125}I was available, then the patient was included in the study. Electrocardiographic monitoring was maintained for at least 3 days.

Detection of deep venous thrombosis On the morning after admission fibrinogen labelled with ^{125}I was injected intravenously. The patients' legs were scanned 1 hour after injection and then daily for 10 days.

Clinical and investigation details A proforma was constructed upon which could be recorded details of the patients' history, examination findings, and investigations. One part of the proforma was completed within 36 hours after radioisotopic injection; the information recorded serves as the 'initial assessment'. A further section of the proforma was completed within 2 days of the patients' discharge from hospital. This 'final assessment' was used to confirm or refute the diagnosis of myocardial infarction which depended upon the occurrence of characteristic electrocardiographic abnormalities and rise in serum levels of aspartate transaminase and lactic dehydrogenase. These enzymes were determined daily for 3 days after admission. Of the 94 patients studied, 5 were rejected as not fulfilling the diagnostic requirements.

Anticoagulants were not used routinely. Some of the patients who developed deep venous thrombosis or features suggesting mural thrombosis in the heart (Simmons *et al.*, 1972) were anticoagulated with heparin and warfarin sodium.

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Definitions for the study

Angina pectoris was defined as exertional chest pain, relieved by rest, occurring regularly for more than one week before admission.

Duration of major infarction pain indicates the duration of the attack of pain which precipitated admission to hospital.

Left ventricular failure was diagnosed on clinical grounds with, in many patients, the aid of a portable chest *x*-ray if there was clinical doubt.

Significant dysrhythmia was judged present if the patient developed (a) atrial fibrillation, (b) supraventricular tachycardia, (c) second-degree or complete heart block, (d) ventricular tachycardia or fibrillation, or (e) ventricular ectopic beats if more than 6 occurred per second, if they were multifocal or arose on any part of the 't' wave.

Results

Of the 89 patients remaining in the study, 24 developed evidence of deep venous thrombosis.

This incidence of 27 per cent agrees broadly with that reported in other studies (Nicolaides *et al.*, 1971; Murray *et al.*, 1970; Maurer *et al.*, 1971; Handley *et al.*, 1972).

Patients were grouped according to sex, age, a previous history of varicose veins, angina pectoris, or myocardial infarction, the site of the present infarct, and whether or not third heart sounds, heart failure, or significant dysrhythmias developed. The incidence of deep venous thrombosis in these various groups is shown in Tables 1 and 2.

The mean duration of major infarct pain in patients developing deep venous thrombosis was 8 hours compared with 8·7 hours in those who did not. This difference is not significant.

The mean of the highest recorded levels of aspartate transaminase and lactic dehydrogenase was 226 units and 2369 units in patients with deep venous thrombosis. These were not significantly different from the means of 219 units, and 2128 units in those without this complication.

Discussion

Statistical analysis of the data reveals that after myocardial infarction deep venous thrombosis is

TABLE 1 *Incidence of deep venous thrombosis under various clinical groupings*

		Patients with deep vein thrombosis	*Patients without deep vein thrombosis*	*Statistical significance*
Sex distribution	Male	18	58	$\chi^2=1·71$
	Female	6	7	Not significant at 95%
History of varicose veins	Present	7	8	$\chi^2=3·76$
	Absent	17	57	Not significant at 95%
Previous myocardial infarction	Present	8	12	$\chi^2=5·76$
	Absent	15	53	Not significant at 95%
	Unknown	1	0	
Previous angina pectoris	Present	10	40	$\chi^2=16·8$
	Absent	13	25	Significant at 95%
	Unknown	1	0	
Site of myocardial infarction	Anterior	13	41	$\chi^2=0·97$
	Inferior	11	23	Not significant
	Unknown	0	1	
Third sound at first assessment	Present	4	11	$\chi^2=0$
	Absent	20	54	Not significant
LV failure	At first assessment: Present	12	17	$\chi^2=4·12$
	At first assessment: Absent	12	48	Significant at 95%
	During admission: Present	15	19	$\chi^2=11·63$
	During admission: Absent	9	46	Significant at 95%
Congestive heart failure	Present	8	8	$\chi^2=7·89$
	Absent	16	57	Not significant at 95%
Significant dysrhythmias	At first assessment: Present	10	14	$\chi^2=4·79$
	At first assessment: Absent	14	51	Significant at 95%
	During admission: Present	12	17	$\chi^2=4·12$
	During admission: Absent	12	48	Significant at 95%

TABLE 2 *Age distribution of patients*

	Patients < 60 years			*Patients > 60 years*		*Unknown*
	30–39	*40–49*	*50–59*	*60–69*	*70–79*	
Patients with deep vein thrombosis	0	4	8	9	2	1
Patients with no deep vein thrombosis	2	21	29	12	1	0
Totals		64		24		1

$\chi^2=8·04$. Significant at 95%.

TABLE 3 *High and low risk groups*

	High risk	*Low risk*
Patients with deep vein thrombosis	23	1
Patients with no deep vein thrombosis	40	25
Totals	63	26

$\chi^2 = 84{\cdot}1$ Significant at 95%

more likely to occur in patients over the age of 60, in those with a previous history of angina pectoris, and in those who develop left or congestive heart failure or significant dysrhythmias. An increased incidence, not reaching statistical significance, was also found in women and patients with a history of varicose veins or previous myocardial infarction.

These results extend the findings of Maurer *et al.* (1971). Using the same method they observed that deep venous thrombosis was more likely to complicate myocardial infarction if the patients had existing varicose veins, were over 70 years old, or had 'a severe complicated illness'. Their findings were not shown to be statistically significant.

In this study duration of pain, site of infarction, the presence of a third heart sound, and the height of the serum enzyme levels had no predictive value.

Patients with one or more statistically significant high risk features were grouped together and their incidence of deep venous thrombosis is compared in Table 3 with that of the remainder.

The difference between the groups is highly significant. It can be inferred from this table that there are certain patients, amounting to 29 per cent of the present series, who are at very low risk of developing deep venous thrombosis. The majority of patients with myocardial infarction can be placed in high or low risk groups at the time of initial assessment. Any steps to prevent or detect deep venous thrombosis can then be concentrated on the patients in the former group.

Whether or not detection of venous thrombosis in these patients leads to a reduction in morbidity or mortality from pulmonary embolism is difficult to assess. In 6 patients there was evidence that thrombosis in calf or popliteal veins was extending. These patients were anticoagulated following the suggestion of Kakkar *et al.* (1969). No patient developed major pulmonary embolism though one patient in whom there was no evidence of deep venous thrombosis died suddenly in his fourth week in hospital. Necropsy was not allowed and it remains possible that he died of major pulmonary embolism.

Three patients were thought to develop minor pulmonary infarction; all had evidence of deep venous thrombosis and none had been anticoagulated. It is well known that minor pulmonary embolism is difficult to diagnose and its true incidence in the patients in this study remains unknown.

It is theoretically possible that intravenous injection of human fibrinogen can transmit serum hepatitis. None of the patients in this study subsequently developed symptomatic hepatitis. One patient developed a rash due to oral potassium iodide given to block thyroid uptake of ^{125}I.

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